=> d his

(FILE 'HOME' ENTERED AT 13:04:43 ON 20 JUN 2003)

FILE 'MEDLINE, BIOSIS' ENTERED AT 13:04:48 ON 20 JUN 2003

L1	291	S	ZUCKER DIABETIC FATTY RAT?
L2	2	S	L1 AND TZD
L3	30	S	L1 AND (PIOGLITAZONE OR ROSIGLITAZONE)
L4	21	DUP REM	L3 (9 DUPLICATES REMOVED)
L5	0	S	L4 AND GLP-1

June 2003

L4 ANSWER 2 OF 21 MEDLINE DUPLICATE 1

TI Intramyocellular lipid and insulin resistance: a longitudinal in vivo 1H-spectroscopic study in **Zucker diabetic fatty rats**.

AU Kuhlmann Johanna; Neumann-Haefelin Claudia; Belz Ulrich; Kalisch Jurgen; Juretschke Hans-Paul; Stein Marion; Kleinschmidt Elke; Kramer Werner; Herling Andreas W

SO DIABETES, (2003 Jan) 52 (1) 138-44.
Journal code: 0372763. ISSN: 0012-1797.

AB Insulin resistance plays an important role in the pathogenesis of human type 2 diabetes. In humans, a negative correlation between insulin sensitivity and intramyocellular lipid (IMCL) content has been shown; thus, IMCL becomes a marker for insulin resistance. Recently, magnetic resonance spectroscopy (MRS) has been established as a dependable method for selective detection and quantification of IMCL in humans. To validate the interrelation between insulin sensitivity and IMCL in an animal model of type 2 diabetes, we established volume selective (1)H-MRS at 7 Tesla to noninvasively assess IMCL in the rat. In male obese **Zucker Diabetic Fatty rats** and their lean littermates, IMCL levels were determined repeatedly over 4 months, and insulin sensitivity was measured by the euglycemic-hyperinsulinemic clamp method at 6-7 and at 22-24 weeks of age. A distinct relation between IMCL and insulin sensitivity was demonstrated as well as age dependence for both parameters. **Rosiglitazone** treatment caused a clear reduction of IMCL and hepatic fat despite increased body weight, and a marked improvement of insulin sensitivity. Thus, the insulin sensitizing properties of **rosiglitazone** were consistent with a redistribution of lipids from nonadipocytic (skeletal muscle, liver) back into fat tissue.